Comparison of endovascular and conventional vascular prostheses in an experimental infection model

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Introduction: **The causes and management of prosthetic graft infections have been extensively studied for conventional bypass grafts; however, the infectivity and therapy for endovascular graft infections are completely unknown. The aim of this study was to compare the biologic properties of infected aortic grafts when inserted by endoluminal or standard transabdominal techniques.**

Methods: **Eighteen dogs underwent placement of polytetrafluoroethylene grafts in their infrarenal aortas either by an endovascular technique (8) or a standard interposition technique (10). Endovascular grafts were constructed from polytetrafluoroethylene (3 cm) and two balloon-expandable stents coaxially mounted onto a balloon catheter delivery system. The grafts were inserted through a left carotid arteriotomy under fluoroscopic control. Initially, seven grafts were infected with decreasing inocula of *Staphylococcus aureus*, starting at 10^7 organisms per ml for 30 minutes and then rinsed briefly (10 seconds) in normal saline solution, until a 50% infective dose for the standard grafts was determined to be 10^2 organisms per ml. After this initial experiment, a second group of 11 dogs were compared at a concentration of 10^2 *S. aureus* per ml. Five dogs underwent endovascular repair, and six dogs had standard graft interpositions after an identical period of bacterial exposure. All grafts were removed at 2 weeks under sterile conditions and were submitted for quantitative culture analysis.**

Results: **Three of the six dogs (50%) with standard grafts appeared to clear their infections, whereas only one of the five dogs (20%) with an endovascular graft was free of organisms at 14 days. This result was further manifested by statistically significant lower postmortem colony counts in the standard grafts ($p < 0.01$).**

Conclusions: **The endoluminal position of the graft and its proximity to the arterial wall do not appear to provide protection against infection. These data suggest that if endovascular grafts become infected, they may be in a disadvantaged position for host defense mechanisms to be effective. (J Vasc Surg 1996;24:920-6.)**

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The management of patients who have an infected aortic prosthesis presents difficult and challenging decisions to the surgeon and is associated with high mortality and amputation rates.[1-4] Graft infection after aortic reconstructive surgery is one of the most disastrous complications in vascular surgery, with mortality rates ranging between 25% to 88%.[5-8] The incidence of limb loss after prosthetic graft infection is 25% to 60%, but this rate can be diminished with ingenious and determined revascularization methods.[9,10] Although the causes and management of these infections have been extensively studied for conventional bypass grafts, the infectivity of endovascular grafts and the therapy for the associated graft infections are completely unknown. Infected intra-

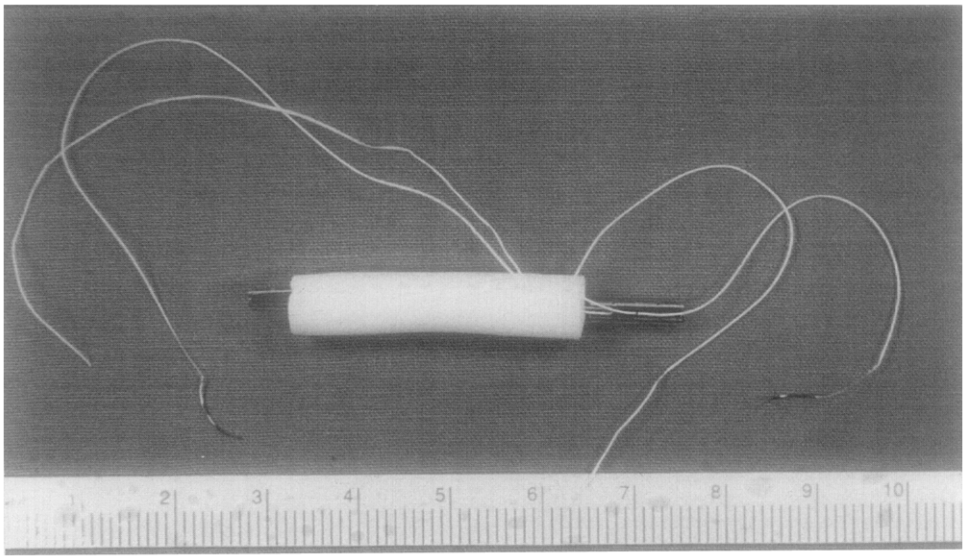

Fig. 1. Suture attachment of PTFE to balloon-expandable stent.

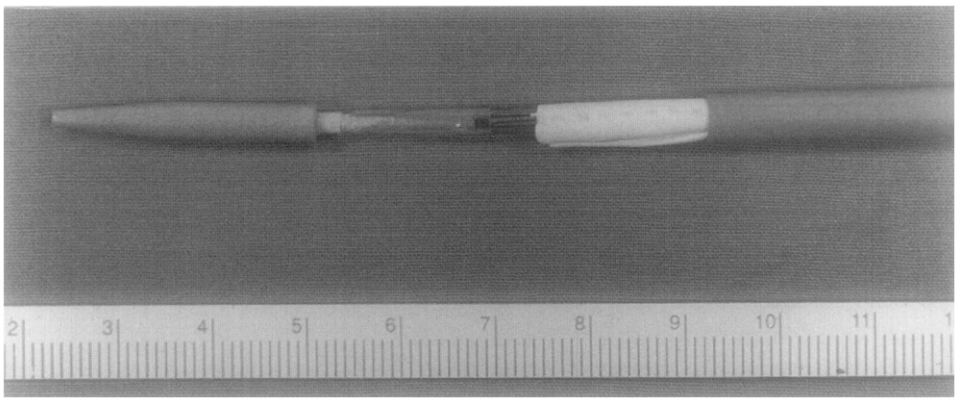

Fig. 2. Coaxially mounted stent-graft is packed into an introducer sheath before sterilization.

vascular stents have been reported,[11] and with the further development and use of endovascular grafts the occurrence of endovascular graft infection is inevitable. The aim of our study was to compare the biologic properties of infected aortic grafts inserted by endoluminal and standard transabdominal techniques. Our hypothesis was that grafts that were placed using endovascular techniques would be more resistant to infection because the immunopotent arterial wall would provide more protection to the outer graft wall when compared with the dead space of the retroperitoneum that is present after standard transabdominal repair.

METHODS

Eighteen female mongrel dogs underwent placement of polytetrafluoroethylene (PTFE) grafts in their infrarenal aortas either by an endovascular (8) or a standard interposition technique (10). Endovascular grafts were assembled from a 3 cm × 6 mm PTFE graft (W.L. Gore and Associates, Flagstaff, Ariz.) and two 1 cm balloon-expandable stents (P104, Johnson and Johnson Interventional Systems, Warren, N.J.) (Fig. 1). The stent-graft combination was coaxially mounted onto a 9 mm × 4 cm Blue Max balloon catheter (Meditech, Inc, Watertown, Mass.) and loaded within an 11F introducer sheath (Meditech) (Fig. 2). The stent-graft combination and delivery system were subjected to conventional gas sterilization with the sheath retracted to allow adequate exposure of the stent-graft combination to the gas (Fig. 2). Standard grafts were constructed by cutting 6-mm PTFE into 3-cm lengths, which were similarly gas-sterilized. While mounted on the balloon cathe-

Table I. Determination of infective dose

No. of dogs	*Inoculum*	*Graft type*	*Systemic signs**	*Temp. >40° C*	*Blood cultures*	*Colony count (× 10^8)*	*Aortic disruption*	*Inflammatory response*	*Survival (days)*
1	10^7	Standard	Septic	Yes	Positive	10	No	Yes	4
1	10^7	Endovascular	Septic	Yes	Positive	10	No	Yes	4
1	10^6	Standard	Septic	Yes	Positive	19	Yes	Yes	5
1	10^4	Standard	Septic	Yes	Positive	90	Yes	Yes	9
1	10^4	Endovascular	Septic	Yes	Positive	45	No	Yes	10
1	10^8	Endovascular	No	No	Negative	17	Yes	No	13
1	10^3	Standard	No	No	Negative	0	No	No	14

*Lethargic, stopped eating.

ter, the endovascular grafts were placed in a test tube of *Staphylococcus aureus* at a known concentration for 30 minutes. The balloon catheter and stent-graft combination were then briefly (10 seconds) rinsed in normal saline solution and retracted into the delivery sheath. The standard PTFE grafts (3 cm) were placed in an identical test tube for 30 minutes, then briefly (10 seconds) rinsed in normal saline solution.

All dogs were treated in accordance with the *Guide for the Care and Use of Laboratory Animals* (NIH Publication No. 86-23, revised 1985). Mongrel dogs weighing between 15 and 25 kg were anesthetized with intravenous sodium pentobarbital (20 mg/kg body weight) and were maintained on Harvard pump ventilators. The dogs were shaved, povidone iodine was applied to the area of the incision three times, and the dogs were sterilely draped. In the endovascular group, a left neck incision was made. The left carotid artery was isolated, and the dogs were systemically heparinized (75 IU/kg body weight) before occlusion of the carotid artery. An arteriotomy was made after ligation of the distal carotid. A 14F introducer sheath (UMI Corp, Ballston Spa, N.Y.) was inserted into the artery, through which an arteriogram was obtained to localize the renal arteries and the aortic trifurcation. A 0.035-inch angiographic wire was threaded into the distal infrarenal aorta under fluoroscopic control. The endovascular device was then placed over the guidewire into the infrarenal aorta, the sheath was withdrawn, and the previously infected graft was deployed by balloon inflation under fluoroscopic control. In the standard group, a midline abdominal incision was made and the infrarenal aorta was exposed, after which the dog was systemically heparinized (75 IU mg/kg body weight), the aorta was clamped, and a 1-cm segment was removed. The previously infected PTFE graft was sutured proximally and distally with running CV-7 PTFE sutures (W.L. Gore and Associates). All dogs received postoperative analgesia (Buprenorphine, 0.2 mg/kg body weight) and had daily body temperatures recorded. Blood samples were taken for culture analysis on postoperative days 1, 7, and 14. All grafts in surviving dogs were removed under sterile conditions at 2 weeks and were submitted for quantitative culture.

RESULTS

Initially, seven grafts were infected with decreasing inocula of *S. aureus*, starting at 10^7 organisms per ml and in decreasing concentrations, until the 50% infective dose for the standard grafts was determined to be 10^2 organisms per ml (Table I). This survivable inoculum was used in all further studies. Findings of comparative gross observations and microbiologic data are shown in Table II.

Three of the six dogs (50%) with standard grafts that were exposed to 10^2 organisms per ml totally cleared their infection, whereas only one of the five dogs with an endovascular graft was free of organisms (20%). This finding was further manifested by a statistically significant lower colony count in the standard grafts ($[2.4 \times 10^8] \pm [2.4 \times 10^8]$) when compared with the endovascular grafts ($[288 \times 10^8] \pm [99 \times 10^8]$) ($p < 0.01$). At an inocula of 10^3 organisms per ml, the closed-space infection in the endovascular graft destroyed the arterial wall and led to rupture, whereas the standard graft was free of organisms at the same concentration. At higher inocula, the host defenses appeared to be overwhelmed in both groups, with no dogs surviving 2 weeks.

DISCUSSION

Resistance of the arterial tree to infection depends on the interaction between the arterial endothelial lining, medial smooth muscle, vasa vasorum, perivascular lymphatics, and the intraluminal blood. Circulating blood in the lumen and vasa vasorum provides the predominant defense against bacterial invasion by providing a continuous source of white blood cells,

Table II. Comparison of endovascular and standard grafts after inoculation with 1×10^2 *S. aureus* per ml

Graft type	*Lethargy/ anorexia*	*Blood cultures positive/total*	*Temp 40° C*	*Colony count*	*Aortic disruption*	*Inflammatory response*	*Survival (days)**
Endovascular	Yes	1/6	No	4.1×10^{10}	Yes	Yes	13
Endovascular	Yes	0/6	No	1.4×10^{10}	No	Yes	14
Endovascular	Yes	1/6	No	3.17×10^{10}	No	Yes	14
Endovascular	Yes	1/6	No	5.7×10^{10}	No	Yes	14
Endovascular	Yes	1/6	No	0	No	No	14
Standard	No	1/6	No	0	No	No	14
Standard	No	1/6	No	1.3×10^{1}	No	No	14
Standard	No	0/6	No	0	No	No	14
Standard	No	0/6	No	0	No	No	14
Standard	No	1/6	No	1.44×10^{9}	Yes	Yes	13
Standard	No	1/6	No	2.1×10^{1}	No	No	14

*Animals killed at 14 days.

antibodies, complement and coagulation proteins. When stimulated by chemotactic factors, white blood cells are able to adhere to endothelial cells and migrate through the endothelium into the deeper layers of the arterial wall to attack bacteria.[12] In the absence of an intact arterial wall, as occurs after standard aortic replacement, the prosthetic arterial graft must rely on the luminal blood flow and perivascular lymphatics without the benefit of the endothelial lining, medial smooth muscle, or vasa vasorum. During endovascular repair these latter defense mechanisms remain operative, and it was our belief that these structures would provide the endovascular graft with an increased resistance to infection.

In this model of aortic graft infection, the bacteria would be at the extraluminal as well as the luminal surface of the prosthetic graft. This model best highlights the differences between endovascular and standard aortic grafts. This was achieved by placing the graft in a known concentration of *S. aureus* for 30 minutes, after which it was briefly rinsed in normal saline solution before implantation. We began with a concentration of 10^7 organisms per ml because this concentration was used in bacteremia models and resulted in a 50% infective dose for standard aortic graft infection in dogs.[13,14] This concentration overwhelmed the dogs in both groups and resulted in systemic sepsis that required that the dogs be killed before the 2-week interval. The concentration of *S. aureus* was gradually reduced. Although the dogs were no longer mortally septic, they began having early aortic disruption until a survivable inocula was achieved (Table I). The first dog to survive the 2-week time period had a standard graft placed with a concentration of 10^3 organisms per ml, and no organisms could be recovered from this prosthetic graft. The comparable dog with an endovascular graft at the same concentration of bacterial inoculation died of an aortic disruption on day 13 with a colony count of 1.7×10^9 organisms recovered from the graft. The concentration was decreased further to 10^2 organisms per ml, which turned out to be the infective dose for 50% of the standard aortic grafts with this model, as half of the dogs completely cleared their grafts of bacteria. Only one of the endovascular grafts was found to be culture-negative at this concentration, and the colony counts in the culture-positive endovascular grafts were significantly higher than those in the standard grafts. This finding implies an increased susceptibility of the host animal to the placement of an infected graft in an endoluminal position. Thus the results of our study suggest that the arterial wall and the vasa vasorum play a limited role in the clearance of aortic graft contamination. The dogs with endovascular grafts showed less resistance to infection, more systemic signs of infection (lethargy, anorexia), and significantly higher colony counts of *S. aureus* when compared with the dogs with standard grafts. The retroperitoneum appears to have a greater ability to deal effectively with bacterial invasion when compared with the arterial wall in a canine model with bacteria, PTFE, and hematoma at the extraluminal surface.

These findings have important implications with regard to the development and increasing use of endovascular devices for the treatment of a variety of vascular lesions.[15] Infection of an endovascular stent with a fatal outcome has recently been reported,[11] and the number of such reports is likely to increase given the increasing use and complexity of endovascular procedures. On the basis of our animal experience, infection of an endovascular graft device may be more virulent and devastating than a standard prosthetic graft infection.

To avoid this difficult complication, the utmost care must be taken to prevent contamination of these devices and their delivery systems. This prevention is facilitated by placement of the graft in a delivery system that allows minimal contact with the patient's skin or operating room personnel. In addition, perioperative antibiotics should be administered and a meticulous, sterile technique maintained.

Bacteria are present in the luminal thrombus of an aneurysm in 14% to 37% of cases, based on bacteriologic studies of aneurysm contents,[16-21] and it is inevitable that the endovascular device will come in contact with these bacteria. Although only 10% or less of these contaminated thrombi lead to graft infection after standard repair, the presence of bacteria at the extraluminal surface of an endovascular graft may be much more significant. The contents of the aneurysm sac, which are in close contact with the prosthetic material, provide a potentially good culture media for bacteria present in the aneurysm wall and are isolated from both antibiotics and white blood cells. If human endovascular grafts are more susceptible to infection, as suggested by this animal model, the incidence of aortic graft infection after endoluminal aortic aneurysm exclusion could be higher than after standard repair.

In our clinical experience there is often a febrile response in the early postoperative period after endoluminal aortic aneurysm repair, particularly in large, clot-filled aneurysms. We believe this may be related to thrombus organization in the aneurysm sac, and to date we have no evidence of endovascular graft infection in our patients; however, our number of cases and follow-up are still quite limited.

It is also still unclear whether these endovascular devices are more susceptible to bacterial seeding during bacteremic episodes and therefore may require prophylactic antibiotics during minor procedures that are associated with bacteremia. The luminal surface would be expected to be similar to other synthetic graft materials; however, there may be other unique properties of an endovascular location that can influence host defenses in response to a bacterial challenge.

CONCLUSION

Endovascular grafts are more susceptible to infection when compared with standard retroperitoneal grafts in a canine model of bacterial seeding of the luminal and extraluminal surface of the graft. Because there is a small amount of blood at the extraluminal surface of the graft in both the endovascular and the standard position, the difference in the infectivity of the two groups may be related to differences in host defenses at the arterial lumen compared with the retroperitoneal position. The vascular wall, in the presence of bacteria, PTFE, and hematoma, does not provide significant protection from a bacterial challenge as compared with the retroperitoneum under similar circumstances. In fact, the retroperitoneal position appears to be superior with regard to resistance to prosthetic graft infection in this setting. This may lead to an increased number or severity of graft infections as the number and complexity of endoluminal procedures expands if these data are confirmed. Further studies to evaluate different graft materials and intravascular systems will be necessary to select the best and safest devices to treat patients.

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DISCUSSION

Dr. Frank T. Padberg (Newark, N.J.). This experiment raises new questions about the biologic behavior of the vascular prosthesis when placed endoluminally, a new procedure in which these authors are clearly in the forefront of development. I would like to congratulate the authors for exploring the risk of infection with this new procedure. Like most good studies, it has raised more questions than it has answered. The results of this investigation were not expected, and they suggest that endovascular prostheses have a higher rate of infection than conventional open-suture implantation. Clearly, one of the expected advantages of the endovascular technique would be the diminished exposure of graft material to infection.

The authors inserted five endovascular and six open arterial grafts of PTFE in canine aortas after exposure to a direct inoculum of 10^2 *S. aureus* per ml for 30 minutes. Two weeks after implantation, the dogs were assessed for systemic signs of sepsis and bacteremia; anastomotic integrity and perigraft inflammation was recorded, and the grafts were submitted for quantitative culture. Systemic signs and colony counts were significantly worse in the endovascular group.

Models of graft infection in canine species produced variable results when based on an intravenous challenge of 1×10^7 colony-forming units of *S. aureus* per ml. Subsequent canine models used a direct inoculum of 1×10^2 *S. aureus* per ml on collagen-coated Dacron grafts. The grafts in the current study were also challenged with a direct inoculum of 10^2 per ml, but the grafts were of PTFE. The decreasing inocula in the initial experiments clearly represents an approximation of a dose-response curve, but the chosen inocula or organism doesn't seem to be quite as severe as the 100% purulent and 100% disincorporation identified in previous studies.

Several questions arise. Is PTFE a greater or lesser risk in this model than if you had performed the endovascular procedures with Dacron?

It is well accepted that incisions in the groin are accompanied by a higher incidence of graft infections. Because this is the usual entry site for human endovascular graft insertion, this position would increase the concern raised from the data presented today. However, note that a recent article reporting the phase 1 experience with the EVT graft reports no graft infections at a mean follow-up of 14 months. Have these findings influenced your clinical conduct of endovascular grafting; and if so, what recommendations might you make for the future in this procedure, as well? What is the incidence of infection in your large clinical series of endovascular grafts?

Experimental investigations with *S. aureus* are appropriate in the assessment of early graft infection such as the 2-week interval chosen here. One wonders, however, whether the same increased susceptibility of the endovascular graft extends to the more indolent infection represented by coagulase-negative *S. epidermidis.* Thus graft type, graft location, the organism, the concentration or inoculum of the organism, and the virulence all represent factors that may influence the infectivity of the vascular prosthesis. Now, in addition, the authors' data suggest that endoluminal placement of PTFE may also increase the risk of graft infection.

Dr. Richard E. Parsons. There are some studies that suggest that PTFE grafts are better than Dacron with regard to infection, but obviously we didn't do that experiment; and I'm not sure whether we would have gotten different results had we done that.

As far as placing these devices through the groin wound, I'm not so concerned about that because the device is in the delivery system, it's sterilized in that system, and the graft comes in minimal contact with the skin or with operating room personnel before it's placed. More concerning to me is the bacteriologic results of culture-positivity of aneurysm contents. When the graft is inflated and it's placed in an aneurysm, it's going to come in contact with these, with fresh blood, prosthetic material, and this may be a problem if these grafts are less resistant.

We have had no clinical incidence of aortic graft infection as yet. We do have some febrile responses, particularly in the large aneurysms. We believe that these responses are related to thrombus organization and have had no experience with one of these grafts becoming infected.

As to the choice of *S. aureus,* it is a fairly common

organism. *S. epidermidis* would have been more indolent and maybe not as dramatic in a short-term study. *S. aureus* was also an organism that our microbiologists had a lot of experience with. This was a virulent pathogen that was recovered from the aortic valve of a patient who died of endocarditis.

Dr. John J. Ricotta (Buffalo, N.Y.). In your open model, is the aorta wrapped back around the prosthesis?

Dr. Parsons. It's not wrapped back around the prosthesis.

Dr. Ricotta. So you wrap retroperitoneum or retroperitoneal tissue over the prosthesis?

Dr. Parsons. Right.

Dr. Ricotta. Do you think that your models are comparable, in other words, your control and your experimental model? Now, when we do aortic aneurysms, they are more comparable because you wrap the aortic sac back around the graft. And it may just be that the aortic wall in fact is sequestering the bacteria, whereas you're exposing the bacteria in your open model to the mesothelium of the retroperitoneum and that is protecting it.

Dr. Parsons. It is interesting, and we didn't expect these results. We thought it was going to be the other way and the endovascular graft was going to be better. The retroperitoneum appears in fact to be better than the aortic wall, and maybe that means that aortic grafts shouldn't be wrapped, I don't really know the answer to that. But it looks like the retroperitoneum is better than the aortic wall. There is a dead space in both. I suppose it's not totally like a clinical model, but I thought it was reasonable and it was very hard to wrap the aorta back around the graft.

Dr. Ricotta. What about the graft do you think makes it more effective?

Dr. Parsons. Well, I think that the graft is totally excluded from the blood and that the aortic wall does not provide much protection to the endothelial surface after it's been excluded from the blood. I think the dead space is the difference.

Dr. Robert P. Leather (Albany, N.Y.). I have one observation, and that is that in our experience with surgical exclusion of the aneurysm, which we have been performing for the past 15 years and have accumulated experience with more than 800 cases, to date we have not had a single case in which there has been infection in this ideal place for infection to grow, namely a big hematoma. So I wouldn't have a great concern about the recovery of bacteria from the contents of the aneurysm. Obviously if this were a significant factor, we should have seen some cases of infection of this ideal culture medium, and we have not. Now, obviously, this doesn't fit the same model you have, although the principles are the same, namely an exclusion rather than a replacement.

Dr. Matthew J. Dougherty (Philadelphia, Pa.). I may have missed it. In your model, did you make an abdominal incision on the dogs that had the endovascular graft?

Dr. Parsons. No, it went through a left neck incision, left carotid.

Dr. Dougherty. I think the results were very interesting, but I wonder whether the lack of infection in the operative group may have more to do with the different response that the animal has to a more major intervention. The immune system may behave somewhat differently in these two groups, and perhaps it would be worthwhile to perform the same study with all animals having at least sham incisions and exposure of the aorta to see whether, in fact, you are dealing with a difference in wall contact as opposed to simply a different type of stress on the animal.

Dr. Parsons. That is interesting. We did not test the stress response. The appealing thing about the endovascular procedure is that it's a smaller operation that should have less complications. And if less response to stress causes you to have more infection, I think that's equally a problem. Maybe the dogs are different than human beings, too.

Dr. Thomas S. Riles (New York, N.Y.). I think maybe one other control would be to actually place the graft in the abdomen and then take it out immediately. It is possible that the manipulations of sewing the graft in and the irrigation of the abdomen and other things we usually do in the operating room decrease the risk of infection of the graft. It may be quite different from a graft that is wrapped up in the sheath and delivered into the aorta. It is my own feeling that there are a lot of processes that may reduce the risk with the open procedure.

Dr. Parsons. Yes, I agree with you. Some modifications of the model may be able to address a variety of additional questions.

Dr. Ricotta. What is the antibiotic prophylaxis that you use clinically now, what drugs do you use? And given this concern, have you changed the antibiotic prophylaxis in terms of either type of drug or duration of drug for your endovascular grafts?

Dr. Parsons. We use Kefzol, and we haven't changed it.